```
$%^STN;HighlightOn= ***;HighlightOff=*** ;


Welcome to STN International! Enter x:x

LOGINID:ssspta1633cxq

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


********** Welcome to STN International **********

 NEWS 1      Web Page URLs for STN Seminar Schedule - N.
America
 NEWS 2 Jan 25 BLAST(R) searching in REGISTRY available in
STN on the Web
 NEWS 3 Jan 29 FSTA has been reloaded and moves to weekly
updates
 NEWS 4 Feb 01 DKILIT now produced by FIZ Karlsruhe and has
a new update
          frequency
 NEWS 5 Feb 19 Access via Tymnet and SprintNet Eliminated
Effective 3/31/02
 NEWS 6 Mar 08 Gene Names now available in BIOSIS
 NEWS 7 Mar 22 TOXLIT no longer available
 NEWS 8 Mar 22 TRCTHERMO no longer available
 NEWS 9 Mar 28 US Provisional Priorities searched with P in
CA/CAplus
          and USPATFULL
 NEWS 10 Mar 28 LIPINSKI/CALC added for property searching in
REGISTRY
 NEWS 11 Apr 02 PAPERCHEM no longer available on STN. Use
PAPERCHEM2 instead.
 NEWS 12 Apr 08 "Ask CAS" for self-help around the clock
 NEWS 13 Apr 09 BEILSTEIN: Reload and Implementation of a
New Subject Area
 NEWS 14 Apr 09 ZDB will be removed from STN
 NEWS 15 Apr 19 US Patent Applications available in IFICDB,
IFIPAT, and IFIUDB
 NEWS 16 Apr 22 Records from IP.com available in CAPLUS,
HCAPLUS, and ZCAPLUS
 NEWS 17 Apr 22 BIOSIS Gene Names now available in
TOXCENTER
 NEWS 18 Apr 22 Federal Research in Progress (FEDRIP) now
available
 NEWS 19 Jun 03 New e-mail delivery for search results now
available
 NEWS 20 Jun 10 MEDLINE Reload
 NEWS 21 Jun 10 PCTFULL has been reloaded
 NEWS 22 Jul 02 FOREGE no longer contains STANDARDS file
segment
 NEWS 23 Jul 19 NTIS to be reloaded July 28, 2002
 NEWS 24 Jul 22 USAN to be reloaded July 28, 2002;
          saved answer sets no longer valid

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS
V6.0d,
          CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND
V6.0Ja(JP),
          AND CURRENT DISCOVER FILE IS DATED 05
FEBRUARY 2002
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network
Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on
that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement. Please note that this agreement limits use to scientific
  research. Use for software development or design or
implementation
  of commercial gateways or other similar uses is prohibited and
may
  result in loss of user privileges and other penalties.

 ********** STN Columbus **********



FILE 'HOME' ENTERED AT 17:26:39 ON 23 JUL 2002

=> FIL BIOSIS EMBASE CAPLUS
COST IN U.S. DOLLARS                SINCE FILE     TOTAL
                                    ENTRY  SESSION
FULL ESTIMATED COST                   0.21      0.21

FILE 'BIOSIS' ENTERED AT 17:26:47 ON 23 JUL 2002
COPYRIGHT (C) 2002 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 17:26:47 ON 23 JUL 2002
COPYRIGHT (C) 2002 Elsevier Science B.V. All rights reserved.

FILE 'CAPLUS' ENTERED AT 17:26:47 ON 23 JUL 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER
AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

=> s endoglin or Alk-2 or activin receptor like kinase or elastin or
Eph4B or EphrinB2
L1      18430 ENDOGLIN OR ALK-2 OR ACTIVIN RECEPTOR
LIKE KINASE OR ELASTIN OR
          EPH4B OR EPHRINB2

=> s endothel? 5(a) remodel?
L2          0 ENDOTHEL? 5(A) REMODEL?

=>
=> s endothel? 5(a) re-model?
L3          0 ENDOTHEL? 5(A) RE-MODEL?

=> s endothel? 3(s) remodel?
L4          6 ENDOTHEL? 3(S) REMODEL?

=> s l1 AND L4
L5          0 L1 AND L4

=> dup rem l4
PROCESSING COMPLETED FOR L4
L6          4 DUP REM L4 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS -
CONTINUE? Y/(N):y

L6  ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2002 ACS
AN  1998:153517  CAPLUS
DN  128:293474
TI  Vascular remodeling and growth factor gene expression in the
rat lung
    during hypoxia
AU  Pfeifer, Michael; Blumberg, Friedrich C.; Wolf, Konrad;
Sandner, Peter;
    Elsner, Dietmar; Riegger, G. unther A. J.; Kurtz, Armin
CS  Klinik und Poliklinik fur Innere Medizin II, University of
Regensburg,
    Franz-Josef-Strauss-Allee 11, Regensburg, D-93042, Germany
SO  Respiration Physiology (1998), 111(2), 201-212
    CODEN: RSPYAK; ISSN: 0034-5687
PB  Elsevier Science B.V.
DT  Journal
LA  English
AB  Recent studies suggest that the vasoactive peptides
endothelin-1 and -3
    and the mitogens VEGF and PDGF-A and -B could be involved
in the
    pathogenesis of hypoxic pulmonary hypertension. Whether
these peptides
    could also be involved in the vascular remodeling occurring
during chronic
    hypoxia (10% oxygen; 1 and 3 wk) was investigated in the rat.
Hypoxia
    increased systolic right ventricular pressure and typical morphol.
signs
    of vascular remodeling were found. This was accompanied by
increased ET-1
    and the ET-3 mRNA expression after acute (6 h) and chronic
hypoxia of 1
    and 3 wk. In contrast, no effects of hypoxia on the gene
expression of
    VEGF and PDGF-A and -B in the lung were found. Thus, ET-3 in
addn. to
    ET-1 could be involved in the process of hypoxia-induced
vascular
    remodeling, whereas it appears less likely that the mitogens
VEGF and
    PDGF-A and -B are essentially involved in the pathogenesis of
hypoxic
    pulmonary hypertension.

L6  ANSWER 2 OF 4  EMBASE  COPYRIGHT 2002 ELSEVIER
SCI. B.V.DUPLICATE 1
AN  1998056433  EMBASE
TI  Carvedilol update IV: Prevention of oxidative stress, cardiac
remodeling
    and progression of congestive heart failure.
AU  Feuerstein G.Z.; Shusterman N.H.; Ruffolo R.R. Jr.
SO  Drugs of Today, (1998) 34/SUPPL. B (1-23).
    Refs: 93
```

ISSN: 0025-7656 CODEN: MDACAP
CY Spain
DT Journal; General Review
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB On May 29, 1997, the United States Food and Drug Administration granted final approval for the use of carvedilol in the treatment of mild to moderate congestive heart failure, in this action, the United States joined 20 countries worldwide that have approved carvedilol (Coreg.RTM./Kredex.RTM.) for treatment of hypertension and congestive heart failure. Carvedilol is also approved for the treatment of angina in several countries. Carvedilol (Fig. 1) is a chemically distinct and pharmacologically unique agent that possesses multiple pharmacological actions, including: 1) nonselective .beta.-adrenoceptor blockade, 2) .alpha.1-adrenoceptor blockade, 3) potent antioxidant activity, and 4) regulation of genes involved in cardiovascular organ ***remodeling*** and apoptosis. Based on this pharmacological profile, carvedilol is uniquely positioned to inhibit several of the major pathological processes that drive the progression of congestive heart failure, including: 1) hemodynamics: reduction of preload, afterload and heart rate; 2) neurohormonal: inhibition of the sympathetic nervous system, renin-angiotensin system and ***endothelin*** ; ***3***) oxidative stress: scavenging potentially toxic oxygen radicals and restoring endogenous antioxidants; 4) genomic reformatting: suppression of several genes associated with pathological organ ***remodeling*** . Thus, carvedilol, through its multiple actions, has the capacity to provide broad cardiovascular organ protection. As a result of these multiple actions, carvedilol, when used in conjunction with standard therapy for heart failure (i.e., diuretics, digoxin, and angiotensin-converting enzyme inhibitors), significantly reduced morbidity, mortality and hospitalization in patients with congestive heart failure of either ischemic or nonischemic (i.e., idiopathic dilated cardiomyopathy) origin, independent of disease severity (mild to moderate) or left ventricular function (ejection fraction). The highly favorable clinical outcomes from the large multicenter clinical trials conducted with carvedilol in the United States and Australia/New Zealand merits a detailed update of the unique mechanisms of action of carvedilol, and a thorough review of the clinical trial results. Accordingly, we will highlight in this update our previous experimental findings with carvedilol as well as more recent data that shed light on the mechanisms by which this drug produces its effects in congestive heart failure. In addition, an update of the results from the large multicenter clinical trials, which formed the basis for the approval of the drug for the treatment of heart failure, will be presented.

L6 ANSWER 3 OF 4 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 2
AN 97289277 EMBASE
DN 1997289277
TI Carvedilol update iv: Prevention of oxidative stress, cardiac remodeling and progression of congestive heart failure.
AU Feuerstein G.Z.; Shusterman N.H.; Ruffolo R.R.
CS R.R. Ruffolo, Pharmacological Sciences, SmithKline Beecham Pharmaceuticals, UW2523, 709 Swedeland Road, King of Prussia, PA 1904-0939, United States
SO Drugs of Today, (1997) 33/7 (453-473).
Refs: 93
ISSN: 0025-7656 CODEN: MDACAP
CY Spain
DT Journal; General Review
FS 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Summary On May 29, 1997, the United States Food and Drug Administration granted final approval for the use of carvedilol in the treatment of mild to moderate congestive heart failure. In this action, the United States joined 20 countries worldwide that have approved carvedilol (Coreg.RTM./Kredex.RTM.) for treatment of hypertension and congestive heart failure. Carvedilol is also approved for the treatment of angina in several countries. Carvedilol (Fig. 1) is a chemically distinct and pharmacologically unique agent that possesses multiple pharmacological actions, including: I)nonselective .beta.-adrenoceptor blockade, 2) .alpha.1-adrenoceptor blockade, 3) potent antioxidant activity, and 4) regulation of genes involved in cardiovascular organ ***remodeling*** and apoptosis. Based on this pharmacological profile, carvedilol is uniquely positioned to inhibit several of the major pathological processes that drive the progression of congestive heart failure, including: 1) hemodynamics: reduction of preload, afterload and heart rate; 2) neurohormonal: inhibition of the sympathetic nervous system, renin-angiotensin system and ***endothelin*** ; ***3***) oxidative stress: scavenging potentially toxic oxygen radicals and restoring endogenous antioxidants; 4) genomic reformatting: suppression of several genes associated with pathological organ ***remodeling*** . Thus, carvedilol, through its multiple actions, has the capacity to provide broad cardiovascular organ protection. As a result of these multiple actions, carvedilol, when used in conjunction with standard therapy for heart failure (i.e., diuretics, digoxin, and angiotensin-converting enzyme inhibitors), significantly reduced morbidity, mortality and hospitalization in patients with congestive heart failure of either ischemic or nonischemic (i.e., idiopathic dilated cardiomyopathy) origin, independent of disease severity (mild to moderate) or left ventricular function (ejection fraction). The highly favorable clinical outcomes from the large multicenter clinical trials conducted with carvedilol in the United States and Australia/New Zealand merits a detailed update of the unique mechanisms of action of carvedilol, and a thorough review of the clinical trial results. Accordingly, we will highlight in this update our previous experimental findings with carvedilol as well as more recent data that shed light on the mechanisms by which this drug produces its effects in congestive heart failure. In addition, an update of the results from the large multicenter clinical trials, which formed the basis for the approval of the drug for the treatment of heart failure, will be presented.

L6 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1993:475290 BIOSIS
DN PREV199396108890
TI Endothelin-1 and endothelin-3 induce chemotaxis and replication of pulmonary artery fibroblasts.
AU Peacock, Andrew J. (1); Dawes, Keith D.; Shock, Anthony; Gray, Andrew J.; Reeves, John T.; Laurent, Geoffrey J.
CS (1) Pulmonary Vascular Unit, Dep. Respiratory Med., Western Infirmary, Glasgow G11 6NT UK
SO American Journal of Respiratory Cell and Molecular Biology, (1992) Vol. 7, No. 5, pp. 492-499.
ISSN: 1044-1549.
DT Article
LA English

AB The ***remodeling*** of pulmonary vessels that occurs in association
with pulmonary hypertension involves, in part, thickening of the adventitia. The stimulus for this process is not understood. One explanation is that endothelial cells secrete a growth factor that expands
the local population of fibroblasts by acting as a chemoattractant and
mitogen. Endothelins are a family of potent newly discovered vasoactive
peptides. One of these compounds, endothelin-1 (ET-1), is secreted by
endothelial cells and is known to constrict pulmonary vessels. Another,
endothelin - ***3*** (ET-3), is not secreted by endothelial
cells and is less potent as a pulmonary vasoconstrictor. We hypothesized
that the endothelins may have the capacity both to constrict these vessels
and to initiate fibroblast chemotaxis and replication. Here we investigated the effects of both ET-1 and ET-3 on the chemotaxis and
replication of fibroblasts derived from pulmonary vessels. Cells were
isolated from rat pulmonary arteries, cultured in medium and 10% newborn
calf serum, and used between passages 2 and 5. Chemotaxis was assessed
using a modified Boyden chamber with a polycarbonate filter (pore size, 8
mu-m) separating cells in the upper chambers from endothelin in the lower
chambers. Replication was assessed both by direct cell counts and by a
colorimetric assay based on uptake and subsequent release of methylene
blue. Both ET-1 and ET-3 induced chemotaxis of pulmonary artery
fibroblasts and did so in a dose-dependent fashion. The maxima for both
peptides occurred at a concentration of about 10-7 M, when chemotaxis was
greatest for ET-1 (22 + 1.4 versus 14 + 1.8 cells/grid (mean + SEM), P
lt 0.05). Both endothelins also induced fibroblast replication, but once
again the effect of ET-1 was greater. Maximal mitogenic response occurred
at a concentration of about 10-5 M and was 35 + 3.1% above control for
ET-1 and 20 + 1% above control for ET-3. These results were obtained in
the presence of 2% newborn calf serum, but results were very similar in
the absence of serum. This study suggests that the endothelins, known to
be vasoconstrictors, also have the capacity to attract fibroblasts and
stimulate their replication. This may have important implications in
pulmonary hypertension, suggesting a possible link between pulmonary
vasoconstriction and an important component of vascular ***remodeling***
.

=> d his

(FILE 'HOME' ENTERED AT 17:26:39 ON 23 JUL 2002)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 17:26:47 ON 23 JUL 2002
L1 18430 S ENDOGLIN OR ALK-2 OR ACTIVIN RECEPTOR LIKE KINASE OR ELASTIN
L2 0 S ENDOTHEL? 5(A) REMODEL?
L3 0 S ENDOTHEL? 5(A) RE-MODEL?
L4 6 S ENDOTHEL? 3(S) REMODEL?
L5 0 S L1 AND L4
L6 4 DUP REM L4 (2 DUPLICATES REMOVED)

=> s l1 5(s) remodel?
MISSING OPERATOR L1 5(S
The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s remodel? 5(s) l1
L7 0 REMODEL? 5(S) L1

=> s l1 and remodel?
L8 542 L1 AND REMODEL?

=> d l8 and vector?
'AND' IS NOT A VALID FORMAT
'VECTOR?' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid in at least one of the files. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.
REENTER DISPLAY FORMAT FOR ALL FILES
(FILEDEFAULT):bib

L8 ANSWER 1 OF 542 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:372650 BIOSIS
DN PREV200200372650
TI In vivo vascular engineering: Directed migration of smooth muscle cells to
limit neointima.
AU Wong, Arthur H.; Waugh, Jacob M.; Amabile, Philippe G.; Yuksel, Eser;
Dake, Michael D. (1)
CS (1) Cardiovascular Interventional Radiology, 300 Pasteur Dr., H3648,
Stanford, CA, 94305: mddake@stanford.edu USA
SO Tissue Engineering, (April, 2002) Vol. 8, No. 2, pp. 189-199.
http://www.liebertpub.com/TEN. print.
ISSN: 1076-3279.
DT Article
LA English

=> s l8 and vector?
L9 6 L8 AND VECTOR?

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 4 DUP REM L9 (2 DUPLICATES REMOVED)

=> d his

(FILE 'HOME' ENTERED AT 17:26:39 ON 23 JUL 2002)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 17:26:47 ON 23 JUL 2002
L1 18430 S ENDOGLIN OR ALK-2 OR ACTIVIN RECEPTOR LIKE KINASE OR ELASTIN
L2 0 S ENDOTHEL? 5(A) REMODEL?
L3 0 S ENDOTHEL? 5(A) RE-MODEL?
L4 6 S ENDOTHEL? 3(S) REMODEL?
L5 0 S L1 AND L4
L6 4 DUP REM L4 (2 DUPLICATES REMOVED)
L7 0 S REMODEL? 5(S) L1
L8 542 S L1 AND REMODEL?
L9 6 S L8 AND VECTOR?
L10 4 DUP REM L9 (2 DUPLICATES REMOVED)

=> s l10 not l6
L11 4 L10 NOT L6

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L11 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:472794 BIOSIS
DN PREV200100472794
TI Identification of a critical Sp1 site within the ***endoglin*** promoter and its involvement in the transforming growth factor-beta
stimulation.
AU Botella, Luisa M.; Sanchez-Elsner, Tilman; Rius, Carlos; Corbi, Angel;
Bernabeu, Carmelo (1)
CS (1) Centro de Investigaciones Biologicas, Consejo Superior de Investigaciones Cientificas, Velazquez 144, 28006, Madrid: bernabeu.c@cib.csic.es Spain
SO Journal of Biological Chemistry, (September 14, 2001) Vol. 276, No. 37,
pp. 34486-34494. print.
ISSN: 0021-9258.
DT Article
LA English
SL English
AB ***Endoglin*** , a component of the transforming growth factor-beta
(TGF-beta) receptor complex expressed on endothelial cells, is involved in
cardiovascular morphogenesis and vascular ***remodeling*** , as
exemplified by the fact that the ***endoglin*** gene is the target for
the autosomal dominant disorder known as hereditary hemorrhagic

telangiectasia type 1. Since haploinsufficiency is the underlying mechanism for hereditary hemorrhagic telangiectasia type 1, understanding the regulation of ***endoglin*** gene expression appears to be a crucial step to correct the disease. In this study we have identified an Sp1 site at -37 as a critical element for the basal transcription of the ***endoglin*** TATA-less promoter. Since ***endoglin*** promoter activity is stimulated by TGF-beta and this stimulation is located at the Sp1-containing proximal region, we have investigated the possible involvement of Sp1 in the TGF-beta-mediated induction. Mutation of the Sp1-binding sequence, or addition of the Sp1 inhibitor WP631, abolished both the basal transcription activity and the TGF-beta responsiveness of the ***endoglin*** promoter. Binding of Sp1 and Smad3 to the proximal promoter region -50/-29 was evidenced by electrophoretic mobility shift assays and DNA affinity precipitation studies. Furthermore, synergistic cooperation on the promoter activity between Sp1 and TGF-beta or Smad3 could be demonstrated by co-transfection experiments of reporter promoter constructs. The molecular mechanism underlying this cooperation appears to involve a direct physical interaction between Sp1 and Smad3/Smad4.

L11 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:237882 BIOSIS
DN PREV200000237882
TI Adenoviral-mediated expression of antisense RNA to basic fibroblast growth factor reduces tangential stress in arterialized vein grafts.
AU Hanna, Abigail K. (1); Duran, Walter N.; Leconte, Isabelle; Fox, Jonathan C.; Neschis, David G.; Hobson, Robert W., II; Golden, Michael A.
CS (1) Section of Vascular Surgery, Yale University School of Medicine, 333 Cedar St, FMB 137, New Haven, CT, 06520-8062 USA
SO Journal of Vascular Surgery, (April, 2000) Vol. 31, No. 4, pp. 770-780. ISSN: 0741-5214.
DT Article
LA English
SL English
AB Purpose: The purpose of this study was to test whether basic fibroblast growth factor (bFGF) participates in arterialized vein graft ***remodeling*** . Methods: Rabbits underwent in vivo gene transfer and carotid interposition vein grafting. Segments of external jugular vein were infected with an adenovirus that expressed antisense bFGF RNA (Ad.ASbFGF) at 1 X 1010 PFU/mL to inhibit new synthesis of bFGF by cells in the vein graft wall. Control rabbits were treated with either adenovirus that encoded beta-galactosidase (Ad.lacZ) at 1 X 1010 PFU/mL or vehicle (phosphate-buffered saline solution (PBS)). At 3 days, 3 grafts per treatment group were harvested for the determination of gene expression of ASbFGF RNA by reverse transcriptase-polymerase chain reaction. Rabbits were killed, and perfusion was fixed 2 months after the grafting. Total wall thickness and lumen circumference of vein grafts and normal arteries were measured in cross sections. Calculated mean tangential stress (+SD) for the ASbFGF-treated group and controls was compared for significance. Grafts were immunohistochemically stained to assess bFGF protein production. Results: Only the grafts infected with the Ad.ASbFGF gene expressed ASbFGF RNA. Grafts that were treated with Ad.ASbFGF displayed lower tangential stress (10.9 + 2.3 dynes/cm2) than PBS alone (22 + 2.8 dynes/cm2) or Ad.lacZ-treated controls (20.6 + 5.4 dynes/cm2; P < .001). Tangential stress in the Ad.ASbFGF group was comparable to a normal carotid artery (13.9 + 2.1 dynes/cm2). The difference in mean total wall thickness was significant among the 3 treatment groups: Ad.ASbFGF, 164 + 3.4 mum); Ad.lacZ, 100 + 3.3 mum; and PBS, 98 + 3.6 mum; P < .01). Luminal circumference was not different among the groups. The Ad.ASbFGF-treated vein graft wall was composed of thick layers of concentric smooth muscle cells and ***elastin*** fibers in contrast to the sponge-like appearance observed in control arterialized vein grafts. Reduction in bFGF protein was noted only in the Ad.ASbFGF-treated group. Conclusion: Inhibition of bFGF synthesis in vivo with the use of adenoviral gene transfer of antisense RNA to bFGF promotes a vein graft with decreased tangential stress while maintaining the luminal area. The vein graft wall is ***remodeled*** and qualitatively resembles an artery so that wall tangential stress in Ad.ASbFGF and normal artery are not significantly different. The lack of significant difference in lumen circumference among groups suggests that wall thickening in the Ad.ASbFGF grafts is not at the expense of luminal narrowing. Our results suggest that ASbFGF RNA expression may represent an effective strategy in limiting the failure of arterialized venous conduits.

L11 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:110959 BIOSIS
DN PREV200000110959
TI Mechanical strain increases type I collagen expression in pulmonary fibroblasts in vitro.
AU Breen, Ellen C. (1)
CS (1) Dept. of Medicine 0623, Univ. of California, San Diego, 9500 Gilman Dr., La Jolla, CA, 92093-0623 USA
SO Journal of Applied Physiology, (Jan., 2000) Vol. 88, No. 1, pp. 203-209. ISSN: 8750-7587.
DT Article
LA English
SL English
AB Tissue ***remodeling*** is an adaptive response to mechanical tension in the lung. However, the role of pulmonary fibroblasts in this response has not been well characterized. This study investigates the influence of extracellular matrix on the response of fibroblasts to mechanical strain. Cells were cultured on flexible-bottom surfaces coated with fibronectin, laminin, or ***elastin*** and exposed to strain. Under these conditions, fibroblasts align perpendicular to the force ***vector*** . This stimulus results in an increase in alpha1(I) procollagen mRNA in cells cultured on laminin or ***elastin*** but not fibronectin. Increased alpha1(I) procollagen mRNA was detected 6 h after exposure to strain and reached control levels by 72 h. (3H)proline incorporation into newly synthesized procollagen reflects changes in mRNA levels. Strained fibroblasts cultured on laminin or ***elastin*** incorporated 190 and 114%, respectively, more (3H)proline into procollagen than did unstrained cells. No difference was detected in strained fibroblasts cultured on fibronectin. These results suggest that fibroblasts respond to mechanical strain in vitro, and this response is signaled by cell-extracellular matrix interactions.

L11 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
AN 2002:493975 CAPLUS
DN 137:52335
TI Manipulation of arterial-venous identity
IN Li, Dean Y.
PA The University of Utah Research Foundation, USA

SO PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002011785	A2	20020214	WO 2001-US24405	20010803
WO 2002011785	A3	20020530		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002081284	A1	20020627	US 2001-921771	20010803

PRAI US 2000-222759P P 20000803

AB Methods and compns. for manipulating the arterial-venous identity of endothelial cells are provided. The methods comprise introducing an arterial mol. program into endothelial cells of a vein section such that the endothelial cells can ***remodel*** to form arterial endothelial cells. The arterial mol. program can comprise one or more polynucleotides encoding various genes that are assocd. with arterial development and/or differentiation from veins. Expression ***vectors*** comprising the genes can be used to introduce the mol. program into the cells. A method of treating a patient having an obstructed blood vessel is also provided.

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	69.53	69.74

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.24	-1.24

STN INTERNATIONAL LOGOFF AT 17:35:51 ON 23 JUL 2002